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ABSTRACT

This study contrasts the performance of a 17-year-old female subject with Turner's syndrome before and after developing left temporal lobe seizures, as a means of identifying the mechanism responsible for the Turner's syndrome spatial impairment. The results revealed a deficit in spatial processing before onset of the seizure disorder. Results also revealed that, after developing the seizure disorder, the subject exhibited a selective verbal recall deficit, a marked improvement in spatial cognitive functioning, and a slight shift towards greater right hemisphere involvement in nonverbal processing. These findings may reflect an interaction between the specific neurological functioning of the patient and the resulting left hemisphere dysfunction from the seizure disorder. The mechanism responsible for the spatial disorder in Turner's syndrome might be one of inappropriate hemisphere activation due to inhibition by the left hemisphere over the right. Includes 45 references. (JDD)

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Improved Spatial Ability Correlated with Left Hemisphere Dysfunction in Turner's Syndrome. Implications for Mechanism.

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ABSTRACT

This study contrasts the performance of a seventeen-year-old female subject with Turner's syndrome (TS) before and after developing left temporal lobe seizures as a means of identifying the mechanism responsible for the TS spatial impairment. The results revealed a deficit in spatial processing before epilepsy and a selective verbal recall deficit, a marked improvement in spatial cognitive functioning and a slight shift towards greater right hemisphere involvement in nonverbal processing after developing the seizure disorder. Improved spatial ability was accounted for by a release from cross-hemisphere interference of the left hemisphere following seizures, thereby allowing the right hemisphere to realize its full potential. An interference model was proposed to account for the spatial deficit in Turner's syndrome.

IMPROVED SPATIAL ABILITY CORRELATED WITH LEFT HEMISPHERE DYSFUNCTION IN TURNER'S SYNDROME. IMPLICATIONS FOR MECHANISM.

INTRODUCTION

Turner's syndrome is a condition which arises from the lack of X chromatin material, most usually a single X chromosome. It results in a number of characteristic physical defects, such as short stature, streak ovaries and webbed necks, (Simpson, 1975), cardiac and renal problems, as well as a selective intellectual deficit for the processing of visuo/spatial information (Shaffer, 1962). This deficit impairs their ability to solve puzzles, read maps and graphs, and do arithmetic (Alexander, Ehrhardt and Money, 1966; Money and Alexander, 1966). Studies have shown that on intelligence tests, individuals with Turner's syndrome typically score 10 to 15 points lower in the nonverbal than the verbal scale; verbal intelligence is usually in the normal range (Money, 1963; Shaffer, 1962).

Recently, Turner's syndrome has been the focus of a number of neuropsychologically-oriented investigations aimed at identifying the mechanism responsible for the spatial impairment. This research falls into two major categories. One group of studies has used traditional neuropsychological methods for the assessment of specific neurological impairments. The results of these investigations have been interpreted as indicating generalized (Silbert, Wolff and Lilienthal, 1977) or localized dysfunctions of the right hemisphere (Pennington, Heaton, Karzmask, Pendleton, Lehman, Shucard, 1985), possibly affecting the right parietal (Money, 1973; McGlone, 1985), and frontal lobes (Waber, 1979). Left hemisphere dysfunctions have also been

implicated (Gordon and Galatzer, 1980). A specific right hemisphere dysfunction has been supported by recent histocytological evidence reflecting pronounced neuropathological aberrations of atherosclerotic nature in the right temporo-parietal area in one of two TS subjects autopsied; this patient had had a previously diagnosed spatial cognitive disorder (Reske-Nielsen, Christensen and Nielsen, 1982). Although abnormal EEGs have also been reported for some (but not all) TS cases, neither extent nor site of abnormality correlates with degree of intellectual impairment (Buchbaum, Henkin, Christiansen, 1974; Nielsen, Nyborg, Dahl, 1977; Poenaru, Stanesco, Poenaru, Stoian, 1970). Thus, although abnormalities in brain function do appear to occur among TS subjects, a single deficit has not as yet been identified.

The second series of investigations has used tasks assessing lateralized hemispheric asymmetries for the processing of verbal or nonverbal information. Their aims were to determine whether the organization of function is atypical in Turner's syndrome and if so, how it relates to their spatial processing deficit. The general consensus is that TS subjects show less left hemispheric specialization for verbal processing (Gordon and Galatzer, 1980; McGlone, 1985; Netley and Rovet, 1983; Pennington et al, 1985; Waber, 1979), with degree of bilaterality correlated with spatial impairment (Netley and Rovet, 1982) and increased left hemispheric involvement for nonverbal processing (Rovet, 1985). According to Gordon and Galatzer (1980), their test profile is one of greater than normal similarity between hemispheres, both of which resemble functionally those ascribed to the left.

In both types of investigations relatively large groups of Turner's patients have been compared with normal, brain-lesioned or short stature controls. This paper offers an alternate methodology. It involves a single case study of a seventeen-year-old female patient with Turner's syndrome who was assessed for neurocognitive impairments after developing left temporal lobe epilepsy. It was thought that changes in her performance following her seizure disorder might yield relevant information about the mechanism contributing to her impairment in spatial abilities.

MATERIALS AND METHOD

Subject

The subject was a seventeen-year-old right-handed female with a $46,XXq^-$ karyotype and ovarian dysgenesis admitted to hospital following one single generalized tonic/clonic seizure lasting 3-5 minutes generation. Several less severe seizures occurred during her stay in hospital. Parents reported a change in behaviour that began about six months earlier when she appeared to be experiencing intermittent (2/week) episodes of a dream-like state lasting 30-60 seconds with sensations of fear, déjà vu, and memory loss. These findings were thought to suggest subclinical seizure activity of about six months duration.

EEG testing revealed a slight asymmetry on the left side with no definite focus. Slow activity was more prominent on the left than the right with occasional sharp wave forms in the mid-head region. A brain scan was normal. Neurological examination revealed a slight facial asymmetry of her cranial nerves with less movement on the right side of the face. Also noted was a suggestion of left drift in her outstretched arms. A diagnosis of a left-sided seizure disorder was given.

She was treated with 100 mg carbamazepine (Tegretol) three times a day. She sustained a second more massive tonic/clonic seizure one month later. The EEG at this time revealed a slight asymmetry with increased slow activity in the mid-head region and no other abnormality. Her seizures have now been completely controlled by this medication for the past two years.

This patient had been hospitalized twice prior to seizures. At the time of her first admission at age 14 for thyroiditis, a study of her chromosomes was conducted because of her short stature (below the fifth percentile), delayed bone age, and delayed menarche. The results revealed a deletion of the long arm of one of her X chromosomes. A laparoscopy on her second admission at age 15 showed streak ovaries.

She was first assessed psychologically when she was 15.5 years of age, at the time of her second hospital admission as part of a routine evaluation for the consequences of her Turner's syndrome. The second evaluation, which took place when she was 17.3 years of age, three days after her first tonic/clonic seizure, was conducted to determine the impact of the seizure disorder on neurocognitive functioning.

Test

(i) Pre-epilespy

Testing included the Wechsler Intelligence Scale for Children - Revised (Wechsler, 1974); the Wide Range Achievement Test (Jastak, Bijou and Jastak, 1978); two experimental tsks of probed serial recall; and two lateral asymmetry tasks, one assessing left hemisphere functions and the other right hemisphere functions.

The two experimental memory tests tapped the recall of visual or auditory serial information. Visual Probed Serial Recall (VPSR) (Wagner, 1976) required the subject to view in order, seven cards placed face down on the table, showing line drawings of two familiar figures. This was then followed by a target item, which the subject was to locate in the original series by exposing cards until it was found. The task was scored as the mean number of incorrect responses per trial for beginning, middle, and end locations. Auditory Probed Serial Recall (APSR) (Cohen and Netley, 1979) is an auditory analogue of VPSR. It consisted of 12 taped lists of 9 digits, each of which were followed by one of three letters. If the letter was "A", the subject was to repeat the first three digits in the list, "B", the middle three digits and "C", the final three digits. This task was scored in terms of digits recalled per probe location.

The two lateral asymmetry tasks were a dichotic listening task involving verbal stimuli and dichhaptic shape recognition task. The dichotic task was presented via a Sony TC270 tape recorder and Pioneer headphones. The taped stimuli consisted of 30 pairs of stop consonants ("Ba", "Pa", "Ga", and "Ka") presented simultaneously to each ear. The task was to repeat what was heard. The tape was played twice with the earphones revered on the second trial to correct for any lateral response biases. The dichhaptic task (Witelson, 1974), consisting of 8 practice and 10 test trials, involved the simultaneous palpation by each hand of a pair of unseen, unfamiliar wooden shapes. Each trial was followed by a recognition task involving a visual array of six unfamiliar shapes, two of which were the same as those that were felt.

(ii) Post-epilepsy

The test battery at this session included the Weschler Adult Intelligence Scale (W.A.I.S., Wechsler, 1955), the S.R.A. Primary Mental Abilities Verbal Meaning and Spatial Relations test (Thurstone, 1963), the Wechsler Memory Scales (Wechsler, 1945) the Goldman-Fristoe-Woodcock Auditory Memory Tests (Goldman et al, 1970), as well as the VPSR and APSR tasks and five lateral asymmetry measures. The latter included the dichotic stop consonants and dichhaptic tasks as well as a dichotic task with melodies as stimuli and two tachistoscopically presented visual half-field tasks.

Dichotic melodies (Kimura, 1964) required the subject to listen to a pair of simultaneously-played tunes, one to each ear, followed by a selection of four normally-presented melodies from which she was to choose the two that were heard dichotically. This task consisted of two practice and 12 test trials. The earphones were reversed midway during the task to control for any difference in loudness between channels.

The lateral half-field tasks were administered via a Gerbrands 3-Channel projection tachistoscope, which exposed directly to the right or left visual fields stimuli presented on 35mm slides. The subject viewed the stimuli while seated in a chair placed in front of the equipment and approximately 170 cm from the screen, with her head restrained by an ophthalmologic chin rest. She was instructed to tell what she saw of stimuli flashed briefly to the right or left of the X, while continuously fixating on a central "X". The visual angle was 4°, exposure time 10 milliseconds, and interstimulus interval, 5 seconds. In one task, Dots, consisting of random patterns of 3 to 8 dots, the subject was to report the number of dots seen. In the other, Letter Triads, vertically-arranged letter triad combinations were presented and the subject

was to state the letters seen. Both tasks consisted of 4 practice and 20 test trials randomly presented to each visual field.

RESULTS

Abilities

Table I presents the TS subject's Wechsler Intelligence Test scale scores and age-corrected subtest scores and the Wide Range Achievement Test results at the two test sessions. Her nonverbal spatial abilities showed substantial improvement after the seizure disorder. This was evident on all performance subtests, particularly Digit Symbol on which she obtained a perfect score completing it in less than the required time. (This subtest was unfortunately not administered in the first testing session.) Her performance on the verbal subtests, on the other hand, did not change across sessions. Prior to developing epilepsy, she obtained a Verbal IQ of 106 and a Performance IQ of 81. A 25-point difference between scales, which is significant (Wechsler, 1974; Kaufman, 1976), is usually interpreted as suggesting a learning impairment (Rudel, 1976; Smith, Coleman, Dakecki, Davis, 1977). After seizures, her Verbal IQ was 104 and her Performance IQ, 112 (or 102 excluding Digit Symbol). These results were substantiated by her performance on the Primary Mental Abilities subtest results, on which she obtained a Verbal IQ of 100 and a Performance IQ of 111. Her achievement test scores were unaffected by the seizure disorder and did not reflect the improvement in spatial ability. She in fact, did worse in Arithmetic after developing epilepsy.

Insert Table I about here

Memory

These results are presented in Table II. Although a generalized memory impairment was not observed, several selective deficits were noted. Particularly poor were verbatim recall of narrative information (Wechsler Memory Scale - Memory for Passages) and recall of ordered information (G.F.W. - Memory for Sequence). Visual recall was found to be clearly superior to verbal. However, because few recall measures were given prior to epilepsy, it cannot be established whether her good visual recall was due to her improved spatial functioning or whether it was evident prior to the onset of seizures. Her performance on the experimental memory tasks showed a change for the auditory serial recall task APSR only, particularly the recall of beginning-list of primacy information, which was poorer after seizures, and end-list or recency information, which was better. A slight decrease in Digit Span performance (Table I) was also noted following epilepsy.

Insert Table II about here

Laterality

The phi coefficient (Kuhn, 1973) was chosen as the index of lateral asymmetry because it accounts for accuracy determining degree of Lateral bias. Both phi scores and individual right and left side performance scores are given in Table III. Although comparisons are limited for several reasons, including the subjects having been given only two of the five tasks prior to seizures (Footnote), the lack of normative data, and only a single series of tests on a single subject, they do show several interesting phenomena. On the task assessing left hemisphere auditory biases (Dichotic Syllables), a left-sided or right hemisphere advantage was evident after seizure, whereas a

right-ear of left-hemisphere advantage was evident before. On the dichotic task presumed to top right hemisphere specialization, she produced the expected left-sided or right-hemisphere bias after seizures. A slight increase in degree of right-hemisphere bias was noted for the shape recognition task. The two visual-processing tasks, given after epilepsy only, revealed a slight left hemisphere bias for verbal information and no bias for nonverbal.

Insert Table III about here

DISCUSSION

The results of this study indicate a definite improvement in the level of visuo-spatial cognitive functioning in a patient with Turner's syndrome after developing a left hemisphere dyfunction due to a seizure disorder. Also noted were a decrease in left hemisphere specialization for verbal processing, accompanied by stronger right hemisphere biases for nonverbal processing, and selective memory deficits.

Although research on epileptic patients has shown that verbal memory skills, particularly for understanding word meanings (Milberg, Greiffenstein, Lewis, Rourke, 1980) and normative informtion (Glowinski, 1973), may be selectively impaired after left temporal lobe seizures (Milner, 1958), there is no evidence that spatial skills improve following the onset of epilepsy (Dodrill, 1983). If anything, individuals with epilepsy do more poorly in the visuo-spatial area (Black, 1976; Lin, 1979). Thus these findings on this patient with Turnor's syndrome must be attributed to factors other than the effect of seizures themselves.

One possibility is that the present results are simply a consequence of repeat testing over a relatively short period of time. However, because of the magnitude of the difference and the fact that different items were involved (WISC-R versus WAIS) as well as the WAIS data concurring with the PMA test results, it seems unlikely that retesting two years subsequently could account for a selective 20-point improvement on one of the two major IQ scales only.

A second possibility is that the present results are an artifact of having used the WISC-R rior to seizures and the WAIS after. It is known that actual scores may be depressed at the upper age limit of the WISC-R and inflated at the lower age limit of the WAIS. Although to the author's knowledge there are no studies which directly compare performance on the two tests, again it is highly unlikely that this could account for such a sizable difference in scores and, in particular, a perfect performance on Coding.

It is also possible that the present findings were due to the medication she was taking. Although most studies indicate an adverse effect of anticonvulsant medication on cognitive functions, there is some evidence that carbamazepine, which the subject in this study was taking, has the least harmful effects (Dodrill and Troupin, 1977; Dreifuss, 1983; Hamster and Petruch, 1978). Furthermore, most effects are cumulative and occur over the long-term (Corbett, Trimber, Nichol, 1985), not at onset. For example, in a controlled study of the effects of anticonvulsants on nonepileptic volunteers, Trimble, Thompsen and Huppert (1980) reported an association between phenytoin (or Dilantin) levels and impaired cognitive functioning, but not between carbamazepine and ability. Subjects on carbamazepine rated themselves as more active, less tired and less depressed than subjects taking placebo or

phenytoin. Their delayed recall of verbal stimuli was, in fact, better than that of controls, but not their recall of visual. Clearly then, although carbamazepine does not appear to affect intellectual functioning as severely as other anticonvulsants, there is no evidence to support enhanced spatial task performance.

Alternatively, the present findings may reflect an interaction between the specific neurological functioning of this patient with Turner's syndrome and the resulting left hemisphere dysfunction from the seizure disorder. Because the appearance of new or different behaviour patterns after brain injury generally is usually interpreted as the removal of an inhibitory control of one part of the brain over another (Denneberg, 1981, 1984), it is conceivable that the impaired spatial ability in this patient with Turner's syndrome who prior to seizures showed the typical is spatial deficit, is reflecting the removal of inhibitory influence. Because only spatial abilities improved after epilepsy and a shift towards greater right hemisphere involvement was also noted on the laterality tasks (see also McIntyre, Pretchard and Lambroso, 1976), it is possible that the interference in this case was caused by a cross-inhibition effect (Kinsbourne, 1982) of the left hemisphere on the right. When the left hemisphere became damaged from the seizure disorder, right hemisphere inhibition by left was reduced, thereby allowing this hemisphere to realize its full potential. These results therefore suggest that the mechanism responsible for the spatial disorder in Turner's syndrome might be one of inappropriate hemispheric activation due to inhibition by the left hemisphere over the right.

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Footnote

Two months prior to the first tonic/clonic seizure, the patient was seen for additional laterality testing. Four laterality tests were given - two dichotic listening tasks (stop consonants and melodies) and two lateral half-field measures involving verbal (letter triads) and nonverbal (dot enumeration) stimuli. Because of the possibility that her seizure disorder may have been present at this time, given parents' concern over recent change in behaviour regarding memory loss and dream-like states, these data were not included in the pre-epilepsy battery. The results reported as percent correct were: dichotic stop consonants, right ear 43.1, left ear 37.5, phi = .067; dichotic melodies, right ear 78.6, left ear 71.4, phi = -.126; half-field letter triads, RVF 56.7, LVF 53.3, phi = .034; half-field dots, RVF 50, LVF 70, phi = .104.

TABLE I

Psychometric Test Results
Before and After Seizure Disorder

	BEFORE	AFTER
WECHSLER INTELLIGENCE TESTS		
Verbal IQ	106	104
Performance IQ	81	112 (102)[a]
Information	11	10
Similarities	12	12
Arithmetic	7	7
Vocabulary	10	11
Comprehension	11	10
Digit Span	10	9
Picture Completion	7	9
Picture Arrangement	10	11
Block Design	8	9
Object Assembly	4	9
Digit Symbol	-	19
SRA PRIMARY MENTAL ABILITIES (PMA)		
Verbal IQ		100
Performance IQ		111
WIDE RANGE ACHIEVEMENT TEST[b]		
Reading	30	25
Spelling	37	42
Arithmetic	21	9

Note: The Wechsler test given before seizures was the WISC-R given at 15 years and after seizures, the WAIS given at 17.3 years;
[a]Value in parentheses, excludes Coding; [b]Reported values are percentiles.

TABLE II

Memory Test Results

	BEFORE	AFTER
WECHSLER MEMORY SCALE		
Memory Quotient		93
Raw Scores:		
1. Information		6
2. Information		4
3. Mental Content		4
4. Memory for Passages		5 (-1 SD)
5. Digits Total		9 (-1 SD)
6. Visual Reproduction		12 (+1 SD)
GOLDMAN FRISTOE WOODCOCK		
Recognition Memory		32
Memory for Content		27
Memory for Sequence		27
VISUAL PROBED SERIAL RECALL		
Beginning	1	1
Middle	0	0
Final	1	1
AUDITORY PROBED SERIAL RECALL		
Beginning	6	3
Middle	4	5
Final	6	11

Note: Goldman-Fristoe-Woodcock test scores are standard scores; Visual Probed Serial Recall scores are mean responses per item; Auditory Probed Serial Recall scores are number of digits correctly recalled (maximum is 12).

TABLE III

Lateral Asymmetry Test Results

	BEFORE			AFTER		
	Right	Left	Phi	Right	Left	Phi
DICHOTIC						
Syllables	51.7	45.0	+.067	65.0	68.3	-.039
Melodies				50.0	63.3	.169
DICHHAPTIC						
Shape Recognition	80.0	90.0	+.140	80.0	100.0	.193
VISUAL HALF FIELD						
Letter Triads				50.0	46.7	.033
Dots				40.0	40.0	.000

Note: Right- and left-side scores are given as the mean percent correct. On Dichotic Syllables and Letter Triads, phi is obtained by subtracting left side scores from right to reflect degree of left hemisphere bias; on Dichotic Melodies, Dichhaptic Shape Recognition and Dots, phi is obtained by subtracting right side scores from left to determine degree of right hemisphere bias.